# PAROTITIS FOLLOWING INJURY OR DISEASE OF THE ABDOMINAL AND PELVIC VISCERA.[1]

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AMONG the many complications which may be met with after operations on or diseases of the abdominal and pelvic viscera, one of the rarest, and at the same time one of the most interesting, is parotitis.

No distinctive name has as yet been given to this variety of parotitis. Secondary parotitis is too wide, as there are other varieties of secondary parotitis than the one now under consideration; postoperative parotitis is too narrow, as a fair number of instances have arisen in the course of disease of the abdominopelvic viscera without operation. The title of this paper, though it is accurate and embraces all the cases, is lengthy and cumbrous as a name, so, as the condition practically only follows lesions of the contents or of the wall of the cœlom, this form of parotitis will, for the sake of brevity, be referred to in this paper as cœliac parotitis.

The reports of many of these cases are very imperfect, the occurrence of parotitis being often merely mentioned as an incident in the progress of the case, with no details as to date of onset, side affected, or occurrence of suppuration, etc.; but a sufficient number is reported with enough detail to enable conclusions to be drawn.

The primary lesions which give rise to cœliac parotitis cover a large field, and vary greatly in their nature and severity. In 1886, Stephen Paget[1] published abstracts of some sixty cases which he had collected, and in the following year published a statistical paper[2] based on reports of 101 cases. These he groups according to their cause, as follows:

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10 cases arose after disease or injury of the urinary tract.

18 cases arose after disease or injury of the alimentary canal.

23 cases arose after disease or injury of the abdominal wall, peritoneum, or pelvic cellular tissue.

50 cases arose after disease or injury or temporary derangements of the generative organs.

It will be seen that lesions of the generative organs account for half the cases, and of this proportion over twenty cases—roughly 25 per cent. of the total number—occurred after removal of cystic ovaries. Nearly fifty more cases have been recorded since 1887, and when these are added to Paget's 101 cases, the proportion of cases arising after ovariotomy is somewhat lessened, which is only what might be expected from the greatly increased scope of abdominal surgery in the last fifteen years; but ovarian cysts still easily head the list of the causes of cœliac parotitis. Perforated gastric ulcer seems to come next in relative frequency, while the other cases have arisen after most of the ordinary abdominal operations, more rarely after disease of the various viscera without operation,—appendicitis, puerperal fever, intestinal obstruction, removal of Fallopian tubes, gastric ulcer (without perforation), and acute gastritis, to name but a few of the conditions; while a few cases have arisen after such trivial causes as the passage of a catheter, the insertion of a pessary, a blow on the testicle, menstrual disturbances, and the menopause. It is to this last group of trivial causes that Paget[2] refers when he speaks of "temporary derangement of the generative organs."

The time of appearance of this parotitis varies considerably: it may come on as soon as the day following an operation, or as long as two or more weeks after. The greatest interval was in a case following removal of a huge hydatid of liver, the parotitis appearing fifty-four days after the operation and twenty-three days after the bladder had first been washed out for cystitis.[3] No average time can be fixed for its appearance, but after operations on the pelvic or abdominal viscera the majority of cases have arisen in the first week; while those following delivery or abortion usually appear in the second week.

In about one-third of the cases both parotid glands have been involved, the second following the first at an interval of about twenty-four to forty-eight hours, as in mumps. The bilateral cases are rather more frequent after ovariotomy than after other conditions (36 per cent. against 25 per cent.). When only one gland is affected, the right one suffers more frequently than the left; but there seems to be no relationship between the side operated on and the side affected by parotitis.

The parotitis may end by resolution in a few days, or may go on to suppuration, the latter being rather the more frequent; and Paget[2] points out that suppuration always takes place when the patient is in a septic state. On the other hand, suppuration in the gland sometimes occurs when the seat of operation remains free from any signs of sepsis. Although suppuration is the more frequent ending, it is evident from what has just been said that it is not an essential feature of the condition, but is an accident due to the additional infection of the already inflamed gland; in most cases by a septic state of the blood, in others by the entrance of pyogenic organisms from the mouth *viâ* Stenson's duct.

As a rule, cœliac parotitis does not cause much constitutional disturbance: there is usually but slight rise of temperature, and high temperature or rigors due to the parotitis *per se* are quite exceptional; but considerable pain is common, and the appearance of a parotid bubo a few days after a patient has undergone a severe operation must always be a rather serious complication from its interference with feeding and from the effects of the pain, and in this indirect fashion may be sufficient to turn the balance against the patient. In Paget's[2] series, death occurred in thirty-seven cases; but he is careful to point out that in every instance this was due to the primary disease or operation and not to the parotitis. In one of his cases, however (No. 32 in the series),[1] it appears very probable, from the short account given, that the parotitis was directly responsible for the death of the patient, as the complication appeared six days after an ovariotomy, suppuration occurred, and, spreading beyond the limits of the gland, set up a diffuse cellulitis of the neck. Up to the time of appearance of the parotitis the patient had had no rise of temperature and was apparently doing well. In another case, reported by the present writer,[4] a right-sided parotitis occurred four days after an appendectomy: the inflammation was of a gangrenous type, and was undoubtedly the direct cause of death, as the patient's abdominal condition was perfectly satisfactory.

It will be evident that a study of cœliac parotitis is rendered difficult by the very conditions under which it arises, for the primary condition is usually a serious one, and overshadows and masks symptoms which may be due to the parotitis *per se*. The cases in which the parotitis follows "temporary derangement of the generative organs," though few in number, are therefore of great value, inasmuch as in these the symptoms due to the primary condition are practically nil, the parotitis stands out as the dominant feature, and can be studied with more exactness. A case of this nature has been recently recorded by Dalché,[5] and will be very briefly described here, before proceeding to discuss the probable cause of this parotitis.

The patient, a woman of forty-four, had reached the menopause some months previously. For a year she had been subject to attacks of double parotitis, recurring usually every month, sometimes every six or eight weeks, rarely twice in a month. One parotid would quickly enlarge, and the other follow suit within twenty-four hours. The skin over the swellings was tense, red, and shiny; there was great pain but little or no fever. After two or three days the swellings would disappear as quickly as they came. The tongue was but slightly coated, there was no dryness of the mouth, and the teeth were in good condition.

The main interest of cœliac parotitis lies in its cause, and various theories have been put forward, none of which, however, give a satisfactory explanation of all the cases. The three chief are (1) the pyæmic theory, (2) the oral sepsis theory, and (3) the "reflex" theory.

(1) The first theory is that the parotitis is due to pyæmic infection of the gland,—in the old surgical sense of the term, be it noted, not in the modern and wider sense. This is most

difficult to accept. Five of Paget's cases certainly arose in the course of puerperal pyæmia, but, excluding these, there are considerably over 100 cases in which this parotitis was the only secondary symptom. Paget[2] points out that in true surgical pyæmia a secondary abscess in the parotid is quite an exceptional occurrence; and it is impossible to believe that all these instances of parotitis were really cases of pyæmia in which the parotitis was the sole secondary symptom. Then in nearly half the cases the gland does not suppurate; and there are also a large number in which the operation area remains free from all sign of sepsis. Further, if pyæmic infection be the real cause, parotitis should surely be found arising occasionally as a solitary event after operations on other parts of the body; but such a case has yet to be recorded.

(2) The second theory is that cœliac parotitis is caused by infection of the gland by micro-organisms from the mouth *via* Stenson's duct, and at first sight this seems the obvious explanation. The mouth is always teeming with microbes; a study of the recorded cases makes it evident that in the majority, the patients, from the nature of the primary condition, must, before the onset of the parotitis, have had thickly furred tongues and coated buccal mucosa, with consequent increase in the number of organisms present; while in some cases an additional source of sepsis in the form of carious teeth has been noted. It is tempting to assume that in oral sepsis lies the cause of this parotitis, but a little consideration will show that it can hardly be the sole cause. In the first place, there are the cases following "temporary derangement of the generative organs," in which oral sepsis, or, at any rate, an increase in the oral septicity, can be practically excluded. Against the other cases being due to oral sepsis it may be urged that oral sepsis from carious teeth is only too common, but parotitis is never met with arising from this cause alone; and that in an immense number of cases of acute disease the patients have foul, dry tongues and mouths,—conditions which should be so favorable to the development of parotitis that, if buccal infection be the *causa causans,* parotitis could hardly fail to be a

fairly common occurrence in nearly all acute illnesses; indeed, one would expect it to be almost as common as acute otitis media in scarlet fever. In the typical case described above, such fleeting and repeated attacks of inflammation could scarcely, as Dalché[5] points out, be due solely to the action of buccal organisms. But one of the strongest arguments against this theory is the fact that the submaxillary and sublingual salivary glands are practically never affected, whereas on this theory one would expect to find them involved quite as frequently as the parotids. But, though oral sepsis cannot be the exciting cause, there is little doubt that it is the source of a pyogenic secondary infection of the gland in some of the suppurative cases. Organisms, no doubt, frequently make their way from the mouth to the gland, but, as is the case with other organs communicating by means of a duct with a mucous surface or with the exterior, the cells, when healthy, are able to deal with these intruders; but when their vitality is lowered by previous injury or inflammation, their bactericidal properties are impaired, a *locus minoris resistentiæ* is created, and the organisms are able to gain a hold and produce their specific effect.

(3) The third theory—that cœliac parotitis is due to reflex nervous action—that it is, in fact, a " sympathetic" inflammation, has received considerable support from many writers on the subject. The argument in its favor may be briefly stated thus: the " sympathy" between the parotid glands and the genital organs is shown by the frequent occurrence of orchitis (or more rarely of oöphoritis or mastitis) as a complication of mumps; the fact that parotitis occurs after ovariotomy shows that this sympathetic current may be reversed; therefore secondary parotitis must be a reflex or " sympathetic" phenomenon.

Like the other two theories discussed, this one will not bear critical examination. In the first place, what evidence is there of a sympathetic nervous connection between the parotids and the generative organs? Of experimental proof there is absolutely none, so far as the writer can discover. The clinical evidence rests chiefly on the occurrence of orchitis in mumps, though the salivation of pregnancy and the enlargement of the salivary glands met with in some of the lower animals during the breeding season are also adduced in support of the theory. One writer[6] on the subject says that the fact that orchitis occurs as often as once in every sixty cases of mumps is proof beyond doubt of a nerve connection between the parotids and the generative organs; another[7] says that "connection between organs as remote as those of the neck and those of the abdomen and pelvis can only exist through the medium of the sympathetic nervous system." One can only conclude that they have never heard of micro-organisms; and wonder if, in acute rheumatism, they regard the involvement of structures as remote from one another as the heart and the ankle- or knee-joints, as proof beyond doubt of a sympathetic nervous connection between them. The orchitis, as well as the other and rarer complications of mumps (meningitis, endocarditis, arthritis, etc.), will surely be more generally regarded as due to the action of the specific micro-organism (or its toxins) which is the cause of mumps, and whose seat of election is the parotid glands. It seems, therefore, that, in the absence of any evidence of a nervous tie between the organs, the reflex theory of the cause of cœliac parotitis breaks down, at any rate as far as the pelvic cases are concerned. In the case of the alimentary canal, however, there is of course a nervous tie with the salivary glands, and the well-known fact that the secretion of saliva may be reflexly influenced by disease or injury of the digestive tract is adduced as evidence in support of this theory. But the undoubted fact that nervous impulses can control secretion is no proof that they can cause inflammation; indeed, all our knowledge of inflammation is opposed to this. It may, however, be urged that reflex action, by temporarily inhibiting the secretion of saliva and so rendering the mouth dry, merely opens a way for the entrance of micro-organisms from the mouth along Stenson's duct; but this is open to the same objections that can be brought against the oral sepsis theory,—that in an enormous number of cases of illness in which there is fever, the same conditions are present, namely, deficient salivary secretion plus a septic state of mouth; but the buccal organisms signally fail to take advantage of the opportunities thus offered them for invading the parotid. Besides, it should be borne in mind that stimuli reaching the gland through the sympathetic cause constriction of the blood-vessels, and so prevent that congestion which is so favorable a predisposing cause to microbic invasion. Again, stimuli reaching the salivary glands from the alimentary canal in all probability affect all three pairs of glands, and we should therefore expect to find the submaxillary and sublingual glands involved fairly often; but, as a matter of fact, the number of cases in which they have been affected is so small as to be negligible. But more important than all is the fact that a secondary parotitis, similar in all respects to the cœliac variety, is met with in certain of the acute specific fevers, in which neither abdominal nor pelvic viscera are affected, and in which, therefore, this reflex action can be excluded.

If neither pyæmia, oral sepsis, nor reflex action be the cause of cœliac parotitis, to what must we look for the explanation of this interesting condition? Dalché,[5] in commenting on his case of climacteric parotitis mentioned above, compares the parotitis with the congestive disturbances of other organs sometimes met with at the menopause, or in association with menstrual disturbances, and, after a cautious discussion of the various theories, none of which he appears to accept, concludes his paper with these words: " One thinks more readily that it arose primarily from vasomotor disturbances, aroused perhaps by a genital autointoxication." The writer believes that this suggestion gives the clue to the cause of all the cases.

Besides the action of the nervous system, a tie between organs remote from one another may be brought about by chemical agency, and in following up Dalché's suggestion it is necessary to see, firstly, if parotitis can be caused by the action of toxic substances; and, secondly, if in cases of cœliac parotitis there is any evidence of toxic substances being present in the blood. It may be said at once that there is evidence in favor of both these points. In the first place, in mumps (to which many cases of cœliac parotitis have a strong resemblance) we have a parotitis due to the action of toxic material of microbic origin. Then there is the secondary parotitis met with in cases of chronic poisoning by lead, mercury, copper, and iodine; and that substances like this can cause a very acute inflammation is shown by a case of parotitis due to poisoning by iodide of potassium mentioned by Comby,[8] in which the swelling of the glands reached a maximum in a few minutes. Lastly, there is the variety of secondary parotitis met with as a complication of certain acute specific fevers, namely, in enteric fever, cholera, typhus, small-pox, pneumonia, cerebrospinal meningitis, and (exceedingly rarely) in scarlet fever. In the first two the alimentary canal is, of course, involved, but in the others neither abdominal nor pelvic viscera are affected, and therefore the reflex theory cannot be invoked. Now it is in some of these diseases that secondary parotitis is most frequently met with: in enteric fever it occurs in 2 per cent. of the cases; in cerebrospinal meningitis it is said to be fairly common, and in typhus (a toxæmic and septicæmic disease *par excellence*) it reaches its greatest frequency, about 5 per cent.; while after abdominal operations its incidence, at the most liberal estimate, cannot be 1 per cent.; and if every case of abdominal and pelvic disease be added, its frequency, of course, is enormously decreased. Now the one condition which characterizes all these widely different specific diseases is that in all of them toxic substances are present in the blood; and the writer submits that it is these toxic substances which are the cause of the parotitis, which appears, as a rule, about the height of the disease, or, in other words, when the toxic material is present in greatest abundance. It will, of course, be objected that the parotitis is due, not to the toxins in the blood, but to the septic state of the mouth which is so frequently present; but that this is not really the case is shown by the fact that parotitis is not met with in other acute infectious diseases, such as acute rheumatism, yellow fever, and diphtheria, in all of which, and especially in the last, the mouth is in a septic state; nor, as already pointed out, is it met with in the numberless other cases of acute disease in which an equally foul condition of the mouth is found. Further, in those infectious diseases in which parotitis occurs, its different frequency in the various diseases can hardly be explained by differences in oral septicity, while it can easily be explained on the assumption that the parotid is more readily affected by the poisons of some of these diseases than of others; and it is noteworthy that in the two diseases which are frequently marked by extremely septic mouth, namely, scarlet fever and diphtheria, parotitis is respectively extremely seldom and never found. It may therefore be taken that the variety of parotitis met with as a complication of many of the acute specific fevers is due to the action on the gland of toxic materials present in the blood; and it will also be seen that the parotid gland is an organ which is very susceptible to the action of a number of different poisons.

As regards the second point, whether there is any evidence of toxic material being present in the blood in cases of cœliac parotitis, it is necessary to examine the records of the cases. These show that over 20 per cent. arose in connection with cystic disease of the ovaries. Now the descriptions given in the older works on gynæcology of the constitutional effects of long-standing cases of ovarian cysts, make it quite clear that toxic materials are formed by and absorbed from them in abundance. Nearly as large a proportion of the cases has occurred in connection with other forms of ovarian disease. Now we know that many of our organs manufacture internal secretions which are capable of exerting considerable influence on other parts of the body, and that these secretions are modified by unhealthy conditions of the organs producing them. That the ovaries, and also the testicles, normally produce such substances is beyond dispute; one has only to study the results of their removal when functionally active to see this; while possibly the swelling of the thyroid often met with at the menstrual periods, and the many congestive disturbances which may occur at the menopause, are instances of the action of the ovarian secretion; and Paget[1] mentions a significant case in which parotitis occurred at several successive months in place of the menstrual period. It seems quite possible that the secretion of these genital glands, modified by injury or disease, and of the ovarian cysts, may be irritating to the parotids, and may cause them to become inflamed. Turning to the alimentary cases, it is noticeable that in half of them peritonitis was present, and we know too well that in this condition there is free absorption of toxic material; while in some cases the bacillus coli may possibly be present in the blood. In the alimentary cases without peritonitis there were, in practically all, such conditions as gastric ulcer, intestinal obstruction or malignant growth in the bowel, in all of which there would consequently have been derangement of digestion and usually increased absorption of intestinal contents. A perusal of the effects of the absorption of intestinal contents given by Bouchard[9] in his work on "Autointoxication" makes it clear that no stretch of imagination is needed to believe that this cause may also be responsible for the production of parotitis; and bearing on this, a case seen by the writer a few years ago is very suggestive, as the parotitis arose in a patient suffering from ptomain poisoning from tainted meat, and in whom local symptoms were very slight, but the constitutional effects of the poison were marked. In the uterine cases it is most probable that the interference with the uterus has a reflex effect upon the ovaries, and so causes a modification of their secretion. This may also be the explanation of the cases arising from puerperal sepsis, or the infection in these cases may have been due to the colon bacillus. The latter view seems to be favored by the fact that, as pointed out above, parotitis does not appear, as a rule, until the second week, which may be due to the septic process in these cases not becoming established so quickly as when it occurs after abdominal section. As to the small remaining group of cases arising after operations on the male bladder, there was cystitis or residual urine in all those of which details are given: the contents of the bladder would in these cases form the source of the toxic material, and probably the slight damage to the mucous membrane caused by the cathetér or sound opened up a path for absorption. This is rather borne out by a case lately related to the writer by a friend, in which parotitis followed a suprapubic prostatectomy.

The writer ventures to hope that enough has been said to show that in all the recorded cases of cœliac parotitis there is actual or presumptive evidence of the presence in the blood of toxic materials: there is, of course, no proof that any of these substances are the cause of the parotitis, but, taking the facts in conjunction with what has been shown of the susceptibility of the parotid gland to various other chemical substances, it is surely justifiable to regard this explanation of the cause of cœliac parotitis as correct.

A few possible objections may now be dealt with. The first is that, if cœliac parotitis be due to toxins, it should always be bilateral, since both glands are equally exposed to the action of the toxins; but this objection has no weight, for in other conditions in which bilateral organs are equally exposed to certain influences, it is quite common for only one to be affected. For instance, in mumps, both testicles are equally exposed to infection, but it is usual for only one to be attacked. Again, in the parotitis due to chronic poisoning by mercury, lead, etc., though it would appear that both glands are always involved, there may be an interval of several weeks between the involvement of the two sides; and in these cases it must be remembered that the action of the poison is continued for a considerable time, while in cœliac parotitis the toxic material has usually but a few days in which to act. Possibly a larger dose or more prolonged action is necessary to affect both sides, and this is rather borne out by the fact that double cœliac parotitis occurs most often in connection with ovarian disease, in which absorption has been going on for a longer period than in most of the other cases.

The next objection is that on this autointoxication theory the parotitis should be a frequent occurrence, but its rarity is more difficult to explain on the other theories discussed. On the oral sepsis theory, one would expect it to be fairly common; on the reflex theory it should be the most frequent complication of abdominal and pelvic diseases; in fact, as Dalché[6] pertinently remarks, "it is strange that these (nerve) connections so seldom exert their influence." The objection is easily overcome by the autointoxication theory. In the first place, one would not expect the parotitis to arise after a simple abdominal section with but little handling of bowel, no soiling of the peritoneum, and no subsequent peritonitis; and so far no case has been reported as following such an operation. In cases in which peritonitis is present, or in which there is disease or injury of secreting organs, but in which parotitis does not occur, it is quite justifiable to assume that the particular toxic materials are not formed or absorbed in sufficient quantity to affect the parotid; or that they are excreted or neutralized before they can affect the gland. Only a few cases of chronic lead-poisoning develop parotitis. No one doubts that post-diphtheritic paralysis is due to the toxins of the diphtheria bacillus, because it only occurs in a small percentage of the cases.

The last objection is best expressed by the query, Why should these toxic substances pick out the parotid glands of all organs and affect them only? To this it can only be answered that the cells of certain organs will react to certain poisons, while the cells of other organs remain unaffected. It is not considered necessary to explain why the toxins of scarlet fever should have such a marked effect upon the kidneys; or, to take a more particular instance, why atropine should pick out and paralyze the endings of the third nerve in the iris.

To sum up:

1. It appears most probable that cœliac parotitis is due to the action on the parotid glands of toxic substances absorbed into the blood and derived from (*a*) the secretions of certain organs modified by injury or disease; (*b*) toxins of microbic origin (*e.g.*, bacillus coli) absorbed either from the alimentary canal, peritoneal cavity, or bladder; (*c*) products of deranged digestion.

2. In any given case of injury or disease of the abdominal or pelvic viscera, the occurrence or not of parotitis will there-

fore depend on the presence and the absorption in sufficient quantity of some of these various toxic agents.

3. Suppuration is not an essential feature of the condition, but is due to the fact that the parotid gland, when inflamed by the action of these toxic agents, forms a *locus minoris resistentiæ,* and becomes secondarily infected by pyogenic organisms reaching it (*a*) by the blood-stream; (*b*) by Stenson's duct.

In conclusion, while this theory, like the others discussed, is not capable of proof at the present time, the writer ventures to submit that it is one which affords an explanation of all the cases, and which is in accordance with our present knowledge of pathology.

## REFERENCES.

[1] Paget. Secondary Inflammation of the Parotid, Lancet, Vol. i, 1886.
[2] Paget. Parotitis after Injury or Disease of the Abdomen or Pelvis, Transactions of Medical Society, 1887.
[3] Robinson. Case of very Large Hydatid Cyst of Liver, Transactions of Clinical Society, Vol. xxx.
[4] Dyball. A Fatal Case of Secondary Parotitis, British Medical Journal, Vol. i, 1904.
[5] Dalché. Parotidite d'Origine génitale à la Ménopause, La Gynécologie, 1903.
[6] Brewis. Parotitis following Operations on the Abdominopelvic Viscera, Transactions of the Edinburgh Obstetrical Society, 1892.
[7] Morley. Parotitis following Abdominal Section, American Gynæcology, 1902.
[8] Comby. Les Parotidites Toxiques, Med. Mod., Paris, 1897.
[9] Bouchard. Autointoxication in Disease. (Translated by Oliver.)